TOBACCO CONTROL
AN INTERNATIONAL JOURNAL

Editorials

Please put out that cigarette, grandpa

In the industrialised nations stroke is a leading cause of physical disability and its international significance is accelerating rapidly as life expectancy increases around the globe.[1] But stroke has been a much neglected Cinderella of cardiovascular disease, for perhaps at least three reasons. Firstly, in Westernised countries stroke is far less common as a cause of death than is its sister condition, coronary heart disease (CHD). Secondly, stroke is so much a disease of the very old—half of its victims are over the age of 75 years—and it is only relatively recently, with the decline in mortality from infectious diseases in developing countries and from CHD in many developed countries, that the population at high risk of cerebrovascular disease (CeVD) has begun to expand rapidly.[2] Finally, the lack of effective medical and surgical treatments for acute cerebrovascular events means that stroke tends to induce professional as well as physical paralysis. By contrast, there has been steady development of new treatments for CHD, ranging from aspirin and β blockers, through coronary artery bypass surgery, to coronary angioplasty, thrombolytic agents and now ACE (angiotensin converting enzyme) inhibitor drugs.

The neglect of CeVD has extended to the examination of the relationships between smoking and stroke, where good evidence of an increased risk of stroke in current smokers took much longer to emerge than the evidence showing that smoking caused heart attacks. For example, the 1983 report on smoking and cardiovascular disease from the United States surgeon general[3] concluded unequivocally that "cigarette smoking is one of the three major independent CHD risk factors" (page 5) but stated only that "numerous prospective mortality studies have shown an association between cigarette smoking and cerebrovascular disease" and that "no consistent dose-response effect has been demonstrated" (page 171). By 1989, the surgeon general had concluded that a causal relationship existed.[4]

This issue of *Tobacco Control* carries a report from Bonita and colleagues in New Zealand of a new study showing strong and graded relationships between risk of stroke and both current active smoking and time since quitting, as well as associations between stroke and passive smoking.[5] A well-conducted, population-based, case-control study, it adds to a growing body of publications on this topic that has emerged from the later 1980s onwards. Futhermore, Bonita *et al* demonstrate that use of a reference group consisting of non-smokers who are not exposed to environmental tobacco smoke (ETS) suggests that smoking increases the risk of stroke by as much as six times compared with non-smokers. This is among the highest estimates of the risk of stroke associated with active smoking, but risks may have changed over time as cigarettes and ways of smoking them have changed. The apparent risk is also larger because Bonita *et al* used a different and more appropriate comparison group than was used in the classic epidemiological studies of active smoking. The change in reference group increases the estimates of relative risk by about 50%. If confirmed, risks of this magnitude would implicate smoking in a large proportion of strokes.

The history of neglect of stroke has repeated itself in relation to ETS. For example, a report reviewing the evidence on passive smoking published by Australia's National Health and Medical Research Council (NHMRC) in November 1997[6] cites 17 studies on passive smoking and CHD but only two on passive smoking and CeVD. Ten of the studies of CHD were cohort investigations, whereas both studies of CeVD used a case-control design. The first of these[7] was small (92 cases), used controls selected from other patients in hospital, and it is not clear from the published report that the standard definition of stroke was used to identify cases. The second study[8] avoided the first two of these pitfalls, but most of the 142 cases had had a transient cerebral ischaemic attack rather than a stroke. A recent supplementary report from this group[9] suggests that ETS at home carries a relative risk of a first-ever ischaemic stroke in lifelong non-smokers of 1.70 (with 95% confidence limits of 0.98 and 2.92). Molgaard *et al*[10] mention that they found no relationship between stroke and passive smoking in a further hospital-based, case-control study not mentioned in the review by Australia's NHMRC. The literature also contains reports from the atherosclerosis risk in communities (ARIC) study demonstrating positive relationships between extent of abnormality in the carotid arteries of non-smokers and residence with a smoker 12–14 years previously[11] and at the time that the carotid ultrasound was performed.[12] The same study has also shown that passive smoking is associated with silent cerebral infarction[13] and with the rate of progression of carotid arterial damage,[14] but has yet to publish data on clinically evident stroke.

Given this background, for Bonita *et al* now to present a large (521 cases of first-ever stroke in people aged 74 years or less), population-based, case-control study of stroke and ETS is a very significant step forward in our knowledge. Their estimate of an excess risk of stroke of 82% caused by ETS among lifelong non-smokers or ex-smokers of at least 10 years' standing is important in terms of public health. In New Zealand, where fewer than 20% of adults now smoke,[15 16] ETS is potentially implicated in one in eight of all strokes before very old age. The estimated risk is consistent with the largest of the previous studies,[9] which was based on 154 lifelong non-smokers who suffered an

ischaemic stroke. Further studies, especially in populations where the prevalence of smoking is higher and where rates of stroke are higher, for example in Asia, are now needed to investigate the generalisability of the findings.

We can be confident that the tobacco industry and its fellow travellers will scrutinise the report from New Zealand very closely. It might be asserted that the risk is different beyond the age of 75, when more than half of strokes occur but for which there are no published data. An early criticism is likely to be that the analyses for ETS included some individuals who admitted smoking more than 10 years previously. There will be charges that a fraction of these people will have covertly continued to smoke and that the results are explained by active smoking. Although this amounts to an admission that tobacco does cause CeVD, and would have led to an *under*estimation of the risks of active smoking in the present paper, it is a well-trodden path in the publications on ETS.[17] In their defence, Bonita *et al* can point to independent evidence of dramatic decreases in the prevalence of smoking in their country[18] and to observational data indicating that the risk of stroke diminishes very quickly after cessation of active smoking.[19]

Here, however, a double bind looms. Given the pace of cessation of smoking in New Zealand and the criterion of exposure to ETS used by Bonita *et al*, namely, exposure to the smoke of one member of the household or one co-worker for more than one year in the preceding ten, much of the exposure that they documented could have occurred earlier rather than later in the relevant period, and, by analogy with the evidence on active smoking, any excess risk associated with ETS should have dissipated quickly.

Next, there may be allusions to problems of publication bias, but in the light of the evidence that stroke is a much underinvestigated condition and that documenting the epidemiology of stroke is particularly challenging,[20] there really are very few teams internationally that are in a position to answer as comprehensively the question addressed by Bonita *et al*. We are also likely to hear that not all relevant potential confounders have been taken into account, an oft repeated charge that at times appears to be bandied about mainly on the principle that if enough mud is thrown, some will stick. Even so, no impartial scientist or public health professional is going to argue that policy should be formulated on the basis of a single observational study, no matter how large and well conducted, because all studies are subject to the play of chance and studies of free-living humans are difficult.

At the same time, the appearance of this report adds to the quality, quantity, and scope of the evidence implicating ETS as a cause of serious disease. Three different sets of consequences are likely to follow. First, this study will add to the general momentum for all public places and workplaces to be made smoke-free. Secondly, it should serve as a warning to health authorities in less developed countries where cigarette smoking is increasing and rates of CeVD often exceed those for CHD, for example in most of Asia. Thirdly, it will focus attention on the question of smoking among older people and on the places where they live.

Traditionally efforts to encourage cessation of smoking have neglected the elderly.[21] Partly this may be rationalised by a misconception that those most sensitive to the adverse consequences of smoking will die early and that smokers who continue smoking into old age are resistant to tobacco's dangers. In fact, as the study of British doctors shows, smoking continues to kill as long as individuals smoke.[22] Partly, it is misguided compassion of the kind, "it's the only pleasure Grandpa has got left, so why not leave him alone?" In reality there have always been good reasons why older people should give up smoking: life will be longer and healthier provided smoking has not already initiated a fatal illness; giving up smoking at any age reinforces a clear message to young people, such as grandchildren, that smoking is no longer the norm; many domestic fires are caused by smoking; and, if the older person should be living in sheltered accommodation, this is someone else's workplace. Now Bonita *et al* have pointed to two further reasons: smoking significantly increases the risk of stroke, both in the smoker and in those around him or her.

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